```
=> d hist

     (FILE 'HOME' ENTERED AT 09:10:35 ON 14 MAY 2003)

     FILE 'REGISTRY' ENTERED AT 09:11:00 ON 14 MAY 2003
L1           320 S NEOMYCIN

     FILE 'MEDLINE, CAPLUS, BIOSIS' ENTERED AT 09:14:44 ON 14 MAY 2003
L2         21562 S L1
L3         28309 S NEOMYCIN OR NEAMINE OR NEOBIOSAMINE
L4         32409 S L2 OR L3
L5          3239 S L4 AND (ANTIVIRAL OR VIRUS)
L6           103 S L5 AND REVERSE (W) TRANSCRIPTASE
L7            90 S L6 AND PY<= 2001
L8            50 DUPLICATE REMOVE L7 (40 DUPLICATES REMOVED)

=> s DNA (w) hybrid
L9         2825 DNA (W) HYBRID

=> s RNA (w) hybrid
L10        2047 RNA (W) HYBRID

=> s l9 or l10
L11        4529 L9 OR L10

=> s l8 and l11
L12           0 L8 AND L11

=> l11 and l4
L11 IS NOT A RECOGNIZED COMMAND
The previous command name entered was not recognized by the system.
For a list of commands available to you in the current file, enter
"HELP COMMANDS" at an arrow prompt (=>).

=> s l11 and l4
L13           8 L11 AND L4

=> duplicate remove l13
DUPLICATE PREFERENCE IS 'MEDLINE, CAPLUS, BIOSIS'
KEEP DUPLICATES FROM MORE THAN ONE FILE?  Y/(N):n
PROCESSING COMPLETED FOR L13
L14            7 DUPLICATE REMOVE L13 (1 DUPLICATE REMOVED)

=> d ibib abs 1-7
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L14 ANSWER 12 OF 30 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:177423 CAPLUS
DOCUMENT NUMBER: 135:40492
TITLE: Aminoglycoside antibiotics, neamine and its derivatives as potent inhibitors for the RNA-protein interactions derived from **HIV**-1 activators
AUTHOR(S): Hamasaki, K.; Ueno, A.
CORPORATE SOURCE: Department of Bioengineering, Graduate School of Bioscience and Biotechnology, Tokyo Institute of Technology, Yokohama, 226-8501, Japan
SOURCE: Bioorganic & Medicinal Chemistry Letters (2001), 11(4), 591-594
CODEN: BMCLE8; ISSN: 0960-894X
PUBLISHER: Elsevier Science Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Neamine derivs. which have an arginine (RN), a pyrene (PCN) and both pyrene and arginine (PRN) have been prepd. and their binding toward the RNA fragments derived from **HIV**-1 activator region, TAR and RRE RNA were examd. Among them, PRN bound either TAR RNA or RRE RNA with equiv. binding affinities as Tat and Rev peptide, resp. Neamine derivs. which have an arginine (RN), a pyrene (PCN) and both pyrene and arginine (PRN) have been prepd. and their binding toward the RNA fragments derived from **HIV**-1 activator region, TAR and RRE RNA was examd. Among them, PRN bound either TAR RNA or RRE RNA with equiv. binding affinities as Tat and Rev peptide, resp.
REFERENCE COUNT: 18 THERE ARE 18 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L14 ANSWER 21 OF 30 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1998:275253 CAPLUS
DOCUMENT NUMBER: 128:316931
TITLE: Binding of Neomycin to the TAR Element of **HIV** -1 RNA Induces Dissociation of Tat Protein by an Allosteric Mechanism
AUTHOR(S): Wang, Shaohui; Huber, Paul W.; Cui, Mei; Czarnik, Anthony W.; Mei, Houng-Yau
CORPORATE SOURCE: Department of Chemistry and Biochemistry, University of Notre Dame, Notre Dame, IN, 46556, USA
SOURCE: Biochemistry (1998), 37(16), 5549-5557
CODEN: BICHAW; ISSN: 0006-2960
PUBLISHER: American Chemical Society
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Neomycin inhibits the binding of Tat-derived peptides to the trans-activating region (TAR) of **HIV**-1 RNA. Kinetic studies reveal that neomycin acts as a noncompetitive inhibitor that can bind to the Tat-TAR complex and increase the rate const. (koff) for dissocn. of the peptide from the RNA. Neomycin effects a conformational change in the structure of TAR that can be detected by CD spectroscopy. The increase in ellipticity measured at 265 nm upon binding of the aminoglycoside is opposite to the decrease seen when Tat peptides bind to the RNA. Thus, the structural transition induced by neomycin is apparently incompatible with the binding of Tat and underlies the inhibitory action of the antibiotic. The binding site for neomycin on TAR was identified in RNase protection expts. and is located in the stem immediately below the three-nucleotide bulge that serves as the primary identity element for Tat. Apparent protection of residues in the bulge by neomycin may represent addnl. contacts to the aminoglycoside, but more likely result from changes in the structure of this region when the ligand binds to the RNA. Binding assays using variants of TAR in which inosine residues were substituted for guanosine residues support the results from the RNase protection expts. Inosine substitutions in the lower stem, but not the upper stem, decrease the binding const. for neomycin by approx. 100-fold. Neither of these variants affected the binding affinity of Tat peptide. In addn., these latter expts. suggest that the aminoglycoside may be located in the minor groove of the stem. This mode of assocn. may be a crit. aspect of neomycin's ability to bind to the Tat-TAR complex and could serve as a guide for the design of other drugs that bind to specific RNA targets as noncompetitive inhibitors.

L14 ANSWER 3 OF 7 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:745118 CAPLUS
DOCUMENT NUMBER: 136:33494
TITLE: **Neomycin**-Induced Hybrid Triplex Formation
AUTHOR(S): Arya, Dev P.; Coffee, R. Lane, Jr.; Charles, I.
CORPORATE SOURCE: Laboratory of Medicinal Chemistry Department of Chemistry, Clemson University, Clemson, SC, 29634, USA
SOURCE: Journal of the American Chemical Society (2001), 123(44), 11093-11094
CODEN: JACSAT; ISSN: 0002-7863
PUBLISHER: American Chemical Society
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Recent work has shown the remarkable ability of aminoglycosides (in particular, **neomycin**) to stabilize RNA and DNA triple helixes. The triplex stabilization effect of **neomycin** was shown to be the highest among all groove binders previously studied (which tend to prefer the duplex structures). In our quest to expand the triplex stabilization potential of **neomycin**, we report the remarkable ability of **neomycin** to induce hybrid DNA-RNA-DNA as well as DNA-RNA-RNA triplexes. Aminoglycosides are also shown to stabilize the hybrid DNA-RNA duplex.
REFERENCE COUNT: 32 THERE ARE 32 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 13 OF 26 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1999:502766 CAPLUS
DOCUMENT NUMBER: 131:153725
TITLE: Small molecule inhibition of RNA/ligand binding
INVENTOR(S): Green, Michael R.; Zapp, Maria L.
PATENT ASSIGNEE(S): University of Massachusetts Medical Center, USA
SOURCE: U.S., 14 pp.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 5935776	A	19990810	US 1995-399378	19950302
US 5534408	A	19960709	US 1993-126236	19930924
PRIORITY APPLN. INFO.:			US 1992-965341	19921023
			US 1993-126236	19930924

AB A method is disclosed for the inhibition of binding of a ligand to an RNA, the inhibition being mediated by a small org. mol. that binds to the RNA, thereby inhibiting ligand binding. The invention is particularly directed to the interaction of the Rev protein of HIV with the Rev-responsive element (RRE) present in HIV-derived mRNA mols. A preferred class of small org. mols. are compds. exemplified by 2,5-Bis[4-(2-N,N-dimethylaminopropylamidino)phenyl]furan.

REFERENCE COUNT: 16 THERE ARE 16 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 12 OF 26 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1999:736476 CAPLUS
DOCUMENT NUMBER: 131:346535
TITLE: Use of neomycin for treating angiogenesis-related diseases
INVENTOR(S): Hu, Guo-Fu; Vallee, Bert L.
PATENT ASSIGNEE(S): The Endowment for Research In Human Biology, Inc., USA
SOURCE: PCT Int. Appl., 74 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9958126	A1	19991118	WO 1999-US10269	19990511
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
CA 2331620	AA	19991118	CA 1999-2331620	19990511
AU 9939804	A1	19991129	AU 1999-39804	19990511
EP 1083896	A1	20010321	EP 1999-922915	19990511
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
US 6482802	B1	20021119	US 2000-700436	20001109

PRIORITY APPLN. INFO.: US 1998-84921P P 19980511
WO 1999-US10269 W 19990511

AB The present invention is directed to using neomycin or an analog thereof as a therapeutic agent to treat angiogenesis-related diseases, which are characterized by excessive, undesired or inappropriate angiogenesis or proliferation of endothelial cells. The present invention is also directed to pharmaceutical compns. comprising: (a) neomycin or an analog and, optionally, (b) another anti-angiogenic agent or an anti-neoplastic agent. The present invention is further directed to a method for screening neomycin analogs having anti-angiogenic activity. A preferred embodiment of the invention relates to using neomycin to treat subjects having such diseases. A dose of 20 ng neomycin/embryo or higher completely inhibited angiogenin-induced angiogenesis in the chorioallantoic membrane (CAM) assay. Neomycin inhibits angiogenin-induced angiogenesis mainly through inhibition of nuclear translocation of angiogenin.

REFERENCE COUNT: 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L14 ANSWER 9 OF 30 MEDLINE

ACCESSION NUMBER: 2001609883 MEDLINE

DOCUMENT NUMBER: 21540632 PubMed ID: 11684311

TITLE: Anti-**HIV** activity of a novel aminoglycoside-arginine conjugate.

AUTHOR: Cabrera Cecilia; Gutierrez Arantxa; Barretina Jordi; Blanco Julia; Litovchick Alexander; Lapidot Aviva; Clotet Bonaventura; Este Jose A

CORPORATE SOURCE: Retrovirology Laboratory, Fundacio irsiCaixa, Hospital Universitari Germans Trias i Pujol, Universitat Autonoma de Barcelona, 08916, Badalona, Spain.

SOURCE: ANTIVIRAL RESEARCH, (2002 Jan) 53 (1) 1-8. Journal code: 8109699. ISSN: 0166-3542.

PUB. COUNTRY: Netherlands

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 200203

ENTRY DATE: Entered STN: 20011102
Last Updated on STN: 20020307
Entered Medline: 20020305

AB We have previously described conjugates of L-arginine with aminoglycosides (AAC) that have shown anti-human immunodeficiency virus type 1 (**HIV**-1) activity in in vitro cell culture systems. Here, we extend our report to a novel neomycin B-arginine conjugate (NeoR) that has shown up to 30-fold increased potency over previous AAC compounds. NeoR inhibited the replication of both R5 and X4 strains of **HIV**-1 in cells expressing the appropriate coreceptor or peripheral blood mononuclear cells. In lymphoid tissue ex vivo, NeoR blocked the replication of the dualtropic strain 89.6 suggesting anti-**HIV** activity of AAC on the site of in vivo virus replication. NeoR blocked the binding of **HIV** particles to lymphoid cells and was also able to antagonize the activity of the CXCR4 receptor so it may prevent the emergence of X4 **HIV**-1 strains. Nevertheless, in a cellular assay, we were unable to detect anti-Tat dependent transactivation activity as previously suggested for this family of compounds.

L10 ANSWER 23 OF 26 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1996:408688 BIOSIS
DOCUMENT NUMBER: PREV199699131044
TITLE: Ligands of the antiestrogen-binding site are able to inhibit virion production of human immunodeficiency virus 1-infected lymphocytes.
AUTHOR(S): Mesange, F.; Delarue, F.; Puel, J.; Bayard, F.; Faye, J.-C. (1)
CORPORATE SOURCE: (1) Lab. Endocrinol. Communication Cellulaire, INSERM U397, Inst. Louis Bugnard, CHU Rangueil, Ave. J. Pulhes, 31045 Toulouse Cedex France
SOURCE: Molecular Pharmacology, (1996) Vol. 50, No. 1, pp. 75-79. ISSN: 0026-895X.
DOCUMENT TYPE: Article
LANGUAGE: English
AB Since the discovery of human immunodeficiency retrovirus, the drug arsenal against retrovirus has rapidly increased. Concomitantly, new challenges in the therapy of acquired immune deficiency syndrome have arisen, including drug toxicities, drug resistance, and the development of various cancers as effective therapies prolong survival. Tamoxifen, a nonsteroidal antiestrogen with a low incidence of side effects, is widely used in cancer therapy; it is known to exert pleiotropic activities by binding essentially to the estrogen receptor and other unidentified proteins. In the present work, quantification of the p24 core protein of human immunodeficiency virus 1 produced by infected lymphocytes shows an inhibitory effect of tamoxifen on virion production. Moreover, we assume that this effect is not mediated by the estrogen receptor because antiestrogen ligands interacting with the antiestrogen-binding site exhibit efficacy related to their affinity for this site, although specific antiestrogens of the estrogen receptor are ineffective.